Genetic and environmental origins of health anxiety: a twin study

STEVEN TAYLOR[1], DANA S. THORDARSON[1], KERRY L. JANG[1], GORDON J.G. ASMUNDSON[2]

[1]Department of Psychiatry, University of British Columbia, 2255 Wesbrook Mall, Vancouver, BC, V6T 2A1, Canada
[2]Faculty of Kinesiology and Health Studies, University of Regina, Saskatchewan, S4S OA2, Canada

Excessive health anxiety – which is anxiety about one's health that is disproportionate to the person's medical status – is a common and often debilitating problem. Little is known about its etiology. The present study investigated the role of genetic and environmental factors using a classic twin study method. Results indicated that, after controlling for medical morbidity, environmental influences accounted for most of individual differences in health anxiety. These findings underscore the importance of psychosocial interventions, which have been shown to be among the most effective interventions for excessive health anxiety.

Key words: Health anxiety, genetic factors, environmental factors, psychosocial interventions

Excessive health anxiety is common, often debilitating, and leads to frequent consultations from general medical professionals and mental health specialists. Health anxiety ranges from mild to severe, and is considered maladaptive if it is out of proportion with the person's objective medical status. There are several facets of health anxiety, including health-related fears (i.e., fear that one might succumb to disease, pain, or death), disease conviction (belief that one has a serious disease), excessive health-related behaviors (e.g., needless reassurance-seeking from physicians), and functional impairment (e.g., impairment in occupational functioning because of preoccupation with bodily sensations). When these facets are sufficiently severe and prolonged, the person is likely to meet diagnostic criteria for hypochondriasis (1-4).

Hypochondriasis often arises when the person is under stress, seriously ill, or recovering from a serious illness, or has suffered the loss of a family member (5). Health-anxious people may have a coexisting general medical condition that fuels their anxiety. Psychosocial interventions have been shown to be among the most effective interventions for excessive health anxiety, which suggests that environmental factors play an important role in this disorder. This was supported by Torgersen (6), who found that the concordance rate for lifetime history of somatoform disorders did not significantly differ between monozygotic (MZ) and dizygotic (DZ) twin pairs, despite the two-fold greater genetic similarity of MZ to DZ twins.

A major problem in interpreting Torgersen's results is the very small sample size (N = 35 twin pairs), along with the low population base-rate of hypochondriasis – lifetime prevalence = 1-5% (1) – which would make estimates of concordance in that study highly unreliable.

Larger studies of MZ and DZ twins have examined the heritability of hypochondriasis using the Minnesota Multiphasic Personality Inventory (MMPI) Hypochondriasis (Hs) scale. Results suggested that hypochondriasis is moderately heritable, with genetic factors accounting for up to 35% of the variance in Hs scores (7,8). A problem with these studies was that the MMPI is a poor measure of hypochondriasis, because it assesses awareness of bodily sensations rather than hypochondriasis per se. At best, these studies indicate that one correlate of health anxiety – the tendency to experience recurrent bodily sensations – is moderately heritable.

The goal of the present study was to estimate the relative effect of genetic and environmental factors on the variability of health anxiety measures using a sufficiently large sample and a suitably valid assessment instrument. Unlike any previously published research, this study estimated the heritability of *excessive* health anxiety; that is, the heritability of health anxiety once the effects of medical morbidity were statistically controlled. Prior studies, including studies of patients diagnosed with hypochondriasis, have failed to control for the fact that even many hypochondriacal patients have genuine medical problems that may fuel their health anxiety (4). Many general medical conditions are heritable. Accordingly, in order to investigate the heritability of health anxiety, it is necessary to control for general medical morbidity. Given that health anxiety ranges on a continuum of severity, from mild to full-blown hypochondriasis, we used a general population sample rather than a sample of hypochondriasis patients. A general population sample does not suffer from problems of range restricting in health anxiety scores which would occur if a hypochondriasis sample was used. Range restriction can result in the attenuation of correlations among variables, such as genetic correlations.

METHODS

Participants

The sample consisted of 88 MZ and 65 DZ twin pairs. They were recruited through media announcements for participation in twin research made in major metropolitan centers across Canada, as part of the University of British Columbia Twin Project (9). Most (80%) were female and the mean age was 39 years (SD = 14). Zygosity was determined using an accurate questionnaire (10), along with examina-

tion of recent color photographs. Each twin pair was reared together, and all were fluent in written and spoken English. All participants provided written informed consent, and received an honorarium for completing the study.

Materials and procedure

Twin pairs independently completed a battery of questionnaires in a non-distracting setting at home. This included the Illness Attitude Scales (IAS) (11), which is among the most widely used measures of health anxiety, and has good reliability and validity (4). Factor analytic research indicates that the original nine IAS subscales can be reduced to four subscales, which were used in the present study: intensity of fear of illness, disease, pain, and death; degree of interference in functioning caused by bodily sensations; frequency of treatment-seeking behavior; and strength of disease conviction (12). These dimensions correspond to the major facets of full-blown and subclinical hypochondriasis (1,4). Thus, the IAS assesses a range of health anxiety, from mild to severe.

The IAS also contains sections in which participants are asked to write down any diagnoses and treatments they have obtained from their physicians. In the present study this information was used to gauge each patient's medical morbidity. This was done in an attempt to assess excessive health anxiety separate from any realistic anxiety associated with medically verified diseases. Logistic constraints on data collection did not enable us to obtain data from medical charts or perform physical examinations on the participants. The medical conditions described by the participants were grouped into thirteen categories: gastrointestinal disorders (e.g., ulcerative colitis), cardiovascular diseases (e.g., atrial fibrillation), thyroid conditions (hypo- or hyper-thyroidism), respiratory disorders (e.g., emphysema), gynecological problems (e.g., endometriosis), diabetes, kidney disease, liver disease, neurological disorders (e.g., multiple sclerosis), hematological diseases (e.g., pernicious anemia), chronic pain conditions (e.g., arthritis), chemical sensitivity, and chronic fatigue syndrome. Each participant was given a score of 0 or 1 for each category (absent or present) and the scores were summed across all categories. This likely represents a conservative measure of medical morbidity; it is likely to overestimate to the true severity of medical morbidity because health-anxious people often have the unfounded belief that they have been diagnosed with a general medical condition.

As a check on the validity of our medical morbidity scale, we tested several predictions: scores on this scale should be correlated with age, uncorrelated with gender, correlated with the dimensions of health anxiety (i.e., medical diseases are a source of anxiety), and should be at least moderately heritable (because many diseases have a heritable component). These predictions were largely confirmed. The medical morbidity score was positively correlated with age ($r = .23$, $p<0.01$), uncorrelated with gender (coded such that women = 1, men = 0; $r = -.04$, $p>0.1$), and significantly correlated with the IAS interference, treatment, and conviction dimensions (r ranged from .16 to .20, $p<0.01$) but not with the fear dimension ($r = .07$, $p>0.1$). Scores on the medical morbidity scale were also moderately heritable (heritability = .37). Thus, the scale had acceptable performance on the validity tests.

Statistical procedures

Scores on the medical morbidity scale were partialled out of all scores on the IAS measures of health anxiety. Age and gender were uncorrelated with health anxiety ($r < .10$, $p>0.08$). MZ and DZ within-pair correlations were decomposed via standard biometric structural equation modeling into variance components attributable to additive genetic influences (A), shared environmental influences (C), and nonshared environmental influences (E). This was done by the method of least squares with asymptotic weights using the Mx program (13,14). Additive genetic effects are those that cumulatively influence a given trait (i.e., they are not dominant or epistatic effects). Shared environmental effects are experiences that cause children from the same family to be similar to one another (e.g., a similar standard of living stemming from the parent's income level). Nonshared environmental effects are any experiences that cause children from the same family to be different from one another (e.g., parental favoritism, or an accident to one sibling but not the other). To estimate the degree to which the various dimensions of health anxiety share a common genetic or environmental basis, genetic (r_G) and environmental correlations (r_E) were also computed. They are interpreted in the same way as any other measure of association, such as Pearson's r. To illustrate, the larger the genetic correlation for a pair of variables, the greater the similarity or overlap in the genetic factors that contribute to those variables. If a pair of variables had a large genetic correlation, then that would suggest that they arose from common genetic factors. A low genetic correlation would indicate that they arose from different sets of genes. Environmental correlations are interpreted in the same way.

RESULTS

Table 1 presents the estimates of genetic and environmental influences on individual differences in health anxiety. All scales were modestly heritable, with genetic influences accounting for less than 40% of variability. In contrast, environmental factors accounted for the majority of the variability in the scales (60% to 90%).

Table 2 shows the environmental and genetic correlations among the dimensions of health anxiety. The genetic correlations between scales are generally quite large, suggesting

Table 1 Within-pair correlations, model fit statistics, and parameter estimates for scores on the dimensions of health anxiety (after partialling out the effects of medical morbidity)

			Statistics (χ^2) for fit of model of genetic and environmental effects				Proportion of variance attributable to each effect		
	r_{MZ}	r_{DZ}	ACE	AE	CE	E	h^2	c^2	e^2
Fear of illness, disease, pain, and death	.59	-.03	5.54	**5.54**	10.01	13.59	.37	.00	.63
Interference in functioning caused by bodily sensations	.36	.11	0.26	**0.26**	2.24	12.39	.34	.00	.66
Frequency of treatment-seeking	.19	.02	0.44	**0.44**	0.99	1.70	.13	.00	.87
Disease conviction	.37	-.21	9.36	**9.36**	10.09	10.09	.10	.00	.90

Models fitted using asymptotic weighted least squares. Best-fitting model is in boldface. MZ = monozygotic twins; DZ = dizygotic twins; A = additive genetic effects, C = common (shared) environmental effects, E = non-shared environmental effects, h^2, c^2, and e^2 = proportion of variance due to, respectively, additive genetic factors, common environmental effects, and non-shared environmental effects

Table 2 Genetic correlations (in boldface, above dashes) and nonshared environmental correlations (below dashes) among dimensions of health anxiety (after partialling out medical morbidity)

	Fear of illness, disease, pain, and death	Interference in functioning caused by bodily sensations	Frequency of treatment-seeking	Disease conviction
Fear of illness, disease, pain, and death	-	**.53***	**.44**	**.91***
Interference in functioning caused by bodily sensations	.18*	-	**.69***	**.75**
Frequency of treatment-seeking	.11	.31*	-	**.99**
Disease conviction	.29*	.25*	.05	-

Based on AE models (see Table 1) fitted with asymptotically weighted least square
*p<0.05

that the dimensions of health anxiety are influenced by a common set of genetic factors. Despite their magnitude, not all correlations were statistically significant (this is because the significance of the correlation is a function of the standard errors of the measures). The environmental correlations are much more modest in size, suggesting that most of the environmental influences are dimension specific.

DISCUSSION

The present study is the first to use psychometrically sound measures and a suitably large sample size to investigate the role of genetic and environmental factors in excessive health anxiety (i.e., health anxiety in excess of the person's medical morbidity). Consistent with some of the better earlier research (i.e., the studies using a sufficient sample size, even if those studies used inadequate measures) (7,8), we found evidence that some of the dimensions of health anxiety are moderately heritable. Previous research by DiLalla et al (7) and Gottesman (8) suggested that genetic factors accounted for 35% of variance in health anxiety scores (as assessed by the MMPI Hs scale), whereas the present study indicates that, for the two most heritable dimensions (fears and interference), genetic factors account for 34-37% of variance. The other two dimensions (treatment-seeking and disease conviction) accounted for 10-13% of variance. Our findings are consistent with previous conjectures that health anxiety is largely a learned phenomenon (15,16). The environmental correlations suggest that each dimension of health anxiety arises from a dimension-specific form of environmental (e.g., learning) factor. The nature of these factors remains to be elucidated.

Our findings support the use of environmental interventions, such as psychosocial (cognitive-behavioral) treatments for health anxiety. These are among the most effective interventions (4). Although they appear to be at least as effective as empirically supported pharmacotherapies for health anxiety (e.g., selective serotonin reuptake inhibitors, SSRIs), evidence suggests that the psychosocial interventions have good long-term efficacy, whereas the long-term effects of pharmacotherapies remain to be investigated (4).

The present findings, which suggest that genetic factors play some role in excessive health anxiety, also raise the question of whether pharmacological treatment can be improved by tailoring medications to the person's genotype. Treatment studies for major depression indicate that the efficacy of SSRIs is greater for patients who possess two long alleles of the serotonin transporter gene, compared to patients possessing one or two short alleles (17). The same might be true for excessive health anxiety. More generally, given that excessive health anxiety arises from a combination of genetic and environmental factors, it may be that the optimum treatment involves some combination of pharmacological and psychosocial interventions.

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